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must be satisfied, and inequality (4) implies that $L(v) = 0$. But since $L(u)$ must satisfy equation (6) which is now equivalent to

$$L(u) = \int_a^b u(x)(\beta_1(x) - \beta_2(x))dx + \int_a^b u(x)d(\lambda_1(x) - \lambda_2(x)), \qquad (10)$$

it is necessary that

$$\int_a^b v(x)d(\lambda_1(x) - \lambda_2(x)) = 0.$$

Then equation (9) implies that $\lambda_1(b) - \lambda_2(b) = \lambda_1(a) - \lambda_2(a)$.

If in the above proof the functions $u_n(x)$, $v_j(x)$, and $v(x)$ had been defined as they were on the interval (a, y), where y is an arbitrary point on (a, b), and as identically zero on the rest of (a, b), the same argument would prove that $\lambda_1(y) - \lambda_2(y) = \lambda_1(a) - \lambda_2(a)$. That is, $\lambda_1 - \lambda_2$ is a constant, and equation (10) is equivalent to equation (7), with $\beta = \beta_1 - \beta_2$. This completes the proof of the theorem.

If L also depends on a parameter, y, and if instead of assuming that equation (1) is satisfied for every value of y, it is assumed that $L(u_n(x),y)$ approaches $L(u(x),y)$ in the mean, the theorem does not apply. For instance the functional

$$L(u(x),y) = u(y)$$

satisfies this condition, but cannot be represented by an integral analogous to (7).

[1] Levy, *Bul. Soc. Math. France,* **48,** 1920 (20).

[2] Hart, *New York, Bul. Amer. Math. Soc.,* **27,** 1921 (308).

[3] F. Riesz, *Ann. Sci. Ec. Norm., Paris* (Ser. 3), **31,** 1914 (9–14).

[4] Fréchet, *New York, Trans. Amer. Math. Soc.,* **15,** 1914 (152), or Evans, *Cambridge (Mass.), Colloquium Lectures* (60).

[5] Young, *Proc. London Math. Soc.,* **13,** 1914 (137), or Daniell, *Princeton, Ann. Math.,* **19,** 1917 (288–93).

[6] Bliss, *New York, Bul. Amer. Math. Soc.,* **24,** 1917 (24, corollary).

[7] *Ibid.* (38, Lemma 8).

[8] *Ibid.* (36–40, Theorems, 18 and 19).

SKIN TRANSPLANTATION IN FROG TADPOLES

BY WILLIAM H. COLE

ZOÖLOGICAL LABORATORY, HARVARD UNIVERSITY, AND DEPARTMENT OF BIOLOGY, LAKE FOREST COLLEGE

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1. The experimental inhibition of vision by means of opaque grafts over the eyes of frog tadpoles (from 20 to 100 mm. in length) should contribute evidence as to any regulatory interaction between the graft and the eye, tending to restore the function of the eye. For this purpose 202 operations were performed upon two species of Rana, catesbeiana and clamatans, which had been collected from three widely separated sources.

The first grafts consisted of tail skin placed over the eye. Sixty-six per cent of these showed an adjustment whereby a small part of the graft was absorbed in a manner which tended to restore vision. When back skin was used, no such absorption occurred. Skin from the tail or back, grafted on other parts of the body, persisted indefinitely. However, when tail skin was grafted over hemispheres of glass or celloidin with radii equal to the radius of the eyeball, absorption occurred. The process in these grafts over "artificial eyes" was like that over normal functional eyes. Back-skin grafts so placed were not absorbed. As a check experiment, to determine whether or not the presence of the foreign material caused the absorption, other operations were made in which thin plates of glass cut from the ordinary cover-slip were used instead of the hemispheres. Such grafts were not absorbed, proving that the foreign material was not the cause of absorption.

From a study of the transplants, it was concluded that the curvature of the eyeball and of the artificial eye is the cause of absorption of tail-skin grafts. The plastic tail skin yields to the tension produced by the curvature and adjusts itself. In the majority of cases, this adjustment by absorption of tissue incidentally restores vision. It always ceases as soon as the tension is relieved. When the graft is very thick or is unusually well attached to the host, no adjustment is possible. Nor is it possible in back-skin grafts, because of their more compact structure.

The proliferation of new tissue by the transplants was limited to the tail-skin grafts. In some cases miniature tail tips were formed, while in others irregular proliferation predominated. This latter type is called "amorphic regeneration." The original anterior end, as well as the posterior end, of the graft is able to produce a new notochord and nerve cord in the regenerated tissue. It was found that the absence of light hastens the proliferation period by about nine days, and slightly increases the rate of proliferation. "Dark" grafts regularly showed amorphic regeneration. Following the period of proliferation, all grafts entered a state of equilibrium. In none of the grafts taken from the back or the belly was proliferation observed.

The conclusion is reached that no functional regulation of skin grafts over the eyes occurs. The adjustment which does take place is purely mechanical and its relation to the visual function is quite incidental. The lack of correlative regulation is probably the result of the high state of differentiation and specificity attained by the eye and the skin.

2. When integument from the tail, belly, or back is transplanted to another region on the same animal (auto-transplant), it preserves its characteristic appearance and produces new tissue of its own kind indefinitely. An homoio-transplant (from another individual not closely related) preserves its integrity only temporarily, being ultimately replaced

by regenerated host tissue. From these facts it is concluded that the integument of frog tadpoles is locally specific, and self-differentiating even when transferred to a new soil on the same animal.

3. Auto- and homoio-transplants of white belly skin on the dark back regions always acquire melanophores. The process varies according to the source of the transplant. In auto-transplants there is no antagonistic reaction between the protoplasms of the graft and the host, and union is quickly established. Melanophores appear only after several weeks, sometimes months. They are accompanied by the appearance of an abundance of scattered melanin granules and irregular masses of pigment, which may readily be interpreted as due to the presence of "young" melanophores. In homoio-transplants, union is delayed by the action of the homoio-toxin. In an effort to cover the wound as soon as possible, the surrounding epidermal cells migrate rapidly over the wound, and over the graft as though it were a foreign body. Melanophores are carried along. Such epidermal pigmentation may occur within twenty-four hours. Subsequent increase in the number of epidermal melanophores and the appearance of dermal melanophores and xantholeucophores are caused by formation *in situ* as a result of the normal growth activity of the integument.

It may therefore be said that in auto-transplants the acquisition of melanophores is chiefly the result of formation of pigment from the epithelial cells *in situ,* while in homoio-transplants the pigmentation is chiefly the result of epidermal migration which carries along melanophores. It may be followed by formation *in situ.*

4. When the conjunctiva of frog tadpoles is extensively injured by scratching or pricking, the regenerated tissue is pigmented first by epidermal melanophores and later by dermal melanophores and xantholeucophores. Such pigmentation, which may obscure the eye and presumably impair vision, persists indefinitely. It is further proof that no regulatory action between the eye and the overlying skin exists in tadpoles over 20 millimeters in length. The method of appearance of the pigment throws further light on the question of epidermal wound healing. The extensive injury of the conjunctiva compels the surrounding skin to repair the wound. The epidermal cells migrate over it and carry along the melanophores. Subsequent increase of pigment results from mitotic multiplication of integumentary cells.

5. A series of experiments was performed to discover the stimuli causing expansion and contraction of the dermal melanophores. Briefly, it was found that expansion is caused by a low temperature (near 0° C.) independently of illumination; by darkness; by a 0.1% chloretone solution; by anoxemia and by a low metabolic rate coincident with a moribund state. Contraction of the dermal melanophores is caused by a high temperature (near 35° C.) independently of illumination; by light, and

by a return of normal environmental conditions after removal of the effects of chloretone and anoxemia. Recovery from a moribund state also is accompanied by contraction.

Either expansion or contraction usually requires about thirty-six hours for completion, although the reactions begin within twenty minutes after initiation of the stimulus. Oxygen deficiency brings about complete expansion in about one-half hour. It is probable that any stimulus which lowers the metabolic rate of the whole animal also causes expansion of the melanophores, and any stimulus causing an increase in the metabolic rate results in contraction of the melanophores.

The complete account of these investigations will appear shortly in the *Journal of Experimental Zoölogy.*